Amendments to the claims:

Listing of claims:

1. (Currently amended) A method for determining the predisposition of a human individual to a ~~physiological reaction to~~ variation in glucuronidation activity of a biologically active compound that is metabolized through glucuronidation, said method comprising determining the presence of a polymorphic or haplotypic variation in the nucleotide sequence of exon 1 or the promoter of *UGT1A9* gene ~~or a part thereof~~ of said individual, whereby~~in~~ the presence of the polymorphic or haplotypic variation in said nucleotide sequence is indicative of said predisposition.

2. (Original) The method of claim 1, wherein said predisposition is a hereditary predisposition.

3. (Cancelled) The method of claim 1, wherein said predisposition is selected from the group consisting of a susceptibility, sensibility, diathesis, proneness, proclivity, tendency, sensitivity, responsiveness, resistance and constitutional sickness to said physiological reaction.

4. (Cancelled) The method of claim 1, wherein said physiological reaction is a beneficial reaction.

5. (Currently amended) The method of claim 1, wherein said variation in glucuronidation causes a physiological reaction selected from the group consisting of:~~is at least one of~~ an adverse reaction to said compound, a side effect to said compound, ~~and~~ a variation in response to therapy to said compound, and a modified ability to detoxify food-borne carcinogens.

6. (Cancelled) The method of claim 1, wherein said biologically active compound is a xenobiotic.

7. (Cancelled) The method of claim 6, wherein said xenobiotic is selected from the group consisting of a drug, a carcinogen and a pre-carcinogen.

8. (Currently amended) The method of claim 307, wherein said ~~drug~~ compound is an anti-cancer agent or an immunosuppressive agent.

9. (Original) The method of claim 8, wherein said anti-cancer agent is a camptothecin or an analog thereof.

10. (Previously amended) The method of claim 9, wherein said camptothecin analog is 7-ethyl-10-[4-(1-piperidino)-1-piperidino] carbonyloxy camptothecin (irinotecan, CPT-11), or 7-ethyl-10-hydroxycamptothecin (SN-38).

11. (Original) The method of claim 8, wherein said immunosuppressive agent is mycophenolic acid (MPA).

12. (Cancelled) The method of claim 1, wherein said individual is a human or an animal.

13. (Currently amended) The method of claim 1, wherein said human~~individual~~ has a cancer.

14. (Previously amended) The method of claim 13, wherein said cancer is at least one of colorectal cancer, a solid tumor and a hematological cancer.

15. (Previously amended) The method of claim 1, wherein said determining is performed on a DNA or a RNA sample of said individual.

16. (Cancelled)

17. (Currently amended) The method of claim 1, wherein said polymorphic or haplotypic variation is ~~at least one of a $C^{-2208}T$ substitution, a $C^{-2152}T$ substitution, a $C^{-2141}T$ substitution, a $T^{-1887}G$ substitution, a $T^{-1818}C$ substitution, a $C^{-665}T$ substitution, a $T^{-440}C$ substitution, a $C^{-331}T$ substitution,~~ a $T^{-275}A$ substitution~~, a $G^{-87}A$ substitution, a $G^{8}A$ missence mutation ($C^{3}Y$) and a $T^{98}C$ missence mutation ($M^{33}T$)~~.

18. (Currently amended) The method of claim 3147, wherein said $G^{8}A$ missensee mutation is associated with a decreased predisposition or susceptibility to an anti-cancer agent.

19. (Currently amended) The method of claim 3117, wherein said $G^{8}A$ missensee mutation is associated with a decreased responsiveness to an immunosuppressive agent.

20. (Currently amended) The method of claim 3117, wherein said $T^{98}C$ missensee mutation is associated with an increased adverse reaction to an anti-cancer agent.

21. (Previously amended) The method of claim 1, further comprising determining the presence of a polymorphic or haplotypic variation inis the UGT1A7 gene.

22. (Currently amended) The method of claim 21, wherein said polymorphic or haplotypic variation is at least one of a $G^{353}T$ missense mutation, a $T^{397}G$ missense mutation, a $C^{401}A$ missense mutation, a $G^{402}A$ missense mutations, a $G^{427}C$ missense mutation, and a $T^{632}C$ missense mutation.

23. (Currently amended) The method of claim 21, further comprising determining the presence of a polymorphic or haplotypic variation inis the UGT1A1gene.

24. (Previously amended) The method of claim 23, wherein said polymorphic or haplotypic variation is a TA_7 mutation in the TATA box.

25. (Withdrawn) An isolated nucleotide sequence comprising at least one nucleotide sequence selected from the group consisting of SEQ ID NO: 36, SEQ ID NO: 37, SEQ ID NO: 38, SEQ ID NO: 39, SEQ ID NO: 40, SEQ ID NO: 41, SEQ ID NO: 42, SEQ ID NO: 43, SEQ ID NO: 44, SEQ ID NO: 45 SEQ ID NO: 46, SEQ ID NO: 47, SEQ ID NO: 48, SEQ ID NO: 49, SEQ ID NO: 50, SEQ ID NO: 51, SEQ ID NO: 52, SEQ ID NO: 53, SEQ ID NO: 54, SEQ ID NO: 55, SEQ ID NO: 56, SEQ ID NO: 57, SEQ ID NO: 58, SEQ ID NO: 59, SEQ ID NO: 60, SEQ ID NO: 61, SEQ ID NO: 62, SEQ ID NO: 63, SEQ ID NO: 64, SEQ ID NO: 65, SEQ ID NO: 66, SEQ ID NO: 67, SEQ ID NO: 68, a fragment or the complementary sequences thereof, for determining predisposition to a physiological reaction.

26. (Withdrawn) The nucleotide sequence of claim 25, wherein said sequence is an allelic 6ariant of UGT1A1, UGT1A7 or UGT1A9.

27. (Withdrawn) An isolated amino acid sequence comprising at least one amino acid sequence selected from the group consisting of SEQ ID NO: 69, SEQ ID NO: 70, SEQ ID NO: 71 or a fragment thereof.

28. (Withdrawn) The amino acid sequence of claim 27, wherein said sequence is encoded by a nucleotide sequence comprising at least one sequence selected from the group consisting of SEQ ID NO: 36, SEQ ID NO: 37, SEQ ID NO: 38, a fragment or the complementary sequences thereof.

29. (Withdrawn) The amino acid sequence of claim 27, wherein the expression of said sequence is regulated by a nucleotide sequence comprising at least one sequence selected from the group consisting of SEQ ID NO: 39, SEQ ID NO: 40, SEQ ID NO: 41, SEQ ID NO: 42, SEQ ID NO: 43, SEQ ID NO: 44, SEQ ID NO: 45 SEQ ID NO: 46, SEQ ID NO: 47, SEQ ID NO: 48, SEQ ID NO: 49, SEQ ID NO: 50, SEQ ID NO: 51, SEQ ID NO: 52, SEQ ID NO: 53, SEQ ID NO: 54, SEQ ID NO: 55, SEQ ID NO: 56, SEQ ID NO: 57, SEQ ID NO: 58, SEQ ID NO: 59, a fragment or the complementary sequences thereof.

30. (New) The method according to claim 1, wherein said compound is selected from the group consisting of: an anti-cancer agent, an immunosuppressive agent, a carcinogen and a pre-carcinogen.

31. (New). The method of claim 17, wherein said polymorphic or haplotypic variation is further selected from the groups consisting of: a $C^{-2208}T$ substitution, a $C^{-2152}T$ substitution, a $C^{-2141}T$ substitution, a $T^{-1887}G$ substitution, a $T^{-1818}C$ substitution, a $C^{-665}T$ substitution, a $T^{-440}C$ substitution, a $C^{-331}T$ substitution, a $G^{-87}A$ substitution, a $G^{8}A$ missense mutation ($C^{3}Y$) and a $T^{98}C$ missense mutation ($M^{33}T$).

32. (New) The method of claim 1, wherein said variation in glucuronidation is selected from: a decrease in glucuronidation activity and an increase in glucuronidation activity.